Refine Search

Search Results -

Terms	Documents
L4 and indomethacin	3

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L5

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Thursday, June 29, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L5	L4 and indomethacin	3	L5
L4	cremophor same inflamma$	22	L4
L3	cremophor adj10 inflamma$	0	L3
L2	cremophor	1931	L2
L1	cremophor adj5 inflammation	0	L1

END OF SEARCH HISTORY

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L4: Entry 20 of 22 File: USPT Oct 31, 1995

DOCUMENT-IDENTIFIER: US 5462726 A
TITLE: Method of inhibiting side effects of solvents containing ricinoleic acid or castor oil or derivatives thereof employing a thromboxane A.sub.2 receptor antagonist and pharmaceutical compositions containing such solvents

Detailed Description Text (47):
The following experiments were carried out to examine the effects of cyclosporine A (CsA) and its vehicle, Cremophor EL on force development in isolated vascular smooth muscle and the effects of thromboxane A.sub.2 receptor antagonists, glyburide and BMS 180,291 and the anti-inflammatory agent indomethacin on Cremophor-induced vasoconstriction.

Previous Doc Next Doc Go to Doc#

Refine Search

Search Results -

Terms	Documents
(thromboxane adj3 antagonist) adj5 indomethacin	7

Database:

US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L1

Refine Search

Recall Text | Clear | Interrupt

Search History

DATE: Thursday, June 29, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L1	(thromboxane adj3 antagonist) adj5 indomethacin	7	L1

END OF SEARCH HISTORY

First Hit Previous Doc Next Doc Go to Doc#

Generate Collection Print

L1: Entry 3 of 7 File: DWPI Sep 12, 1986

DERWENT-ACC-NO: 1986-252149
DERWENT-WEEK: 198638

TITLE: Improved absorption of t-PA - by administering hydroxylamine-hydrochloride

Equivalent Abstract Text (2):
Specifically (I) is hydroxylamine or its salts, esp. hydroxylamine hydrochloride (Ia). The TPA and (I) are kept separate before use. The treatment may be combined with electrical stimulation of the injection site to increase muscle blood flow and/or admin. of a platelet aggregation inhibitor, e.g. dazoxiben, thromboxane antagonists SQ27427, aspirin, indomethacin, naproxen or sulfinpyrazone.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
End of Result Set

Generate Collection Print

L5: Entry 1 of 1 File: USPT Jul 13, 1999

DOCUMENT-IDENTIFIER: US 5922690 A
TITLE: Dermatan disulfate, an inhibitor of thrombin generation and activation

Drawing Description Text (4):
FIGS. 3a, 3b, and 3c, show, respectively, graphical representations of the percent compliment inhibition versus concentration by DDS, DS, (parent), DS (commercial), and heparin;

Detailed Description Text (30):
Heparin, DS and DDS were tested for their ability to regulate the classical pathway of complement as previously described and were prepared at various concentrations from 0.15 to 40 .mu.g in 100 .mu.l of half-isotonic veronal-buffered saline, pH 7.5, containing 0.1% gelatin, 0.15M calcium, 0.5 mM magnesium and 2.5% dextrose (DGVB++) and added to tubes on ice. Then, guinea pig C2 (C2gp), pre-titered to produce an average of one hemolytic event per cell (one Z of lysis) was added to each tube in 100 .mu.l of DGVB++. Lastly 1.times.10.sup.7 of sheep erythrocytes that contained surface C1 and C4 (EAC 1,4b) in 100 .mu.l of DGVB++ were added to each tube and the tubes were immediately incubated in a shaking water bath at 30.degree. C. for 10 minutes (the tmax). Then, 0.3 ml guinea pig concentrate (GPC) diluted in gelatin-veronal buffered saline that contained 40 mM ethylene-diamine was added to each tube as a source of terminal complement pathway components and incubation was continued for 60 minutes at 37.degree. C. Finally, 1.5 ml of saline was added to each tube (except the 100% lysis tubes which received water), the tubes were shaken and centrifuged and lysis was assessed by determining hemoglobin release at 414 nm. Tubes that contained no GAG or GAG derivatives were designated as non-inhibited control and were constituted to have about one hemolytic event per cell (one Z of lysis). Reagent blank and 100% lysis tubes received neither GAGs nor C2. Inhibition was calculated based upon the lysis of cellular intermediates (Z) in the test sample compared with the non-inhibited control tubes.

Previous Doc Next Doc Go to Doc#

Refine Search

Search Results -

Terms	Documents
(compliment adj3 activat$) and (antibod$)	8

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L10

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Thursday, June 29, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L10	(compliment adj3 activat$) and (antibod$)	8	L10
L9	(compliment adj3 activat$) and (serine adj1 esterase)	0	L9
L8	(compliment adj3 activat$) and (indomethacin or egta or complestatin or diamine)	0	L8
L7	(compliment ajd3 activat$) and (indomethacin or egta or complestatin or diamine)	31335	L7
L6	compliment$ adj3 activat$	44	L6
L5	L3 and (indomethacin or egta or complestatin or diamine)	1	L5
L4	L3 and (serine adj1 esterase)	0	L4
L3	compliment$ adj3 inhibit$	34	L3
L2	compliment adj2 inhibit$	11	L2
L1	compliment adj1 activation adj1 inhibit$	0	L1

L11: Entry 2 of 11 File: USPT May 13, 2003

DOCUMENT-IDENTIFIER: US 6562343 B1
TITLE: Compositions and methods for the treatment and diagnosis of immune disorders

Detailed Description Text (111):
Fusion proteins, which can facilitate solubility and/or expression, and can increase the blood half-life of the protein, can include, but are not limited to soluble Ig-tailed fusion proteins. Methods for engineering such soluble Ig-tailed fusion proteins are well known to those of skill in the art. See, for example U.S. Pat. No. 5,116,964, which is incorporated herein by reference in its entirety. Further, in addition to the Ig-region encoded by the IgG1 vector, the Fc portion of the Ig region utilized can be modified, by amino acid substitutions, to reduce complement activation and Fc binding. (See, e.g., European Patent No. 239400 B1, Aug. 3, 1994).

Detailed Description Text (117):
Described herein are methods for the production of antibodies capable of specifically recognizing one or more differentially expressed or pathway gene product epitopes. Such antibodies can include, but are not limited to, polyclonal antibodies, monoclonal antibodies (mAbs), humanized or chimeric antibodies, single chain antibodies, Fab fragments, F(ab').sub.2 fragments, fragments produced by a Fab expression library, anti-idiotypic (anti-Id) antibodies, and epitope-binding fragments of any of the above. The Ig tails of such antibodies can be modified to reduce complement activation and Fc binding. (See, for example, European Patent No. 239400 B1, Aug. 3, 1994).

Detailed Description Text (233):
200 gene product peptides corresponding to the ECD having the amino acid sequence shown in FIGS. 17A-17D from about amino acid residue number 21 to about 192 can be used. Human 200 gene product peptides corresponding to the ECD having the amino acid sequence shown in FIGS. 24A-24D from approximately amino acid reside number 21 to about 200. Mutants in which all or part of the hydrophobic anchor sequence (e.g., about amino acid residue number 193 to 214 in FIGS. 17A-17D, or about 201 to about 224 in FIGS. 24A-24D) could also be used. Fusion of these peptides to an IgFc polypeptide should not only increase the stability of the preparation, but will increase the half-life and activity of the fusion protein in vivo. The Fc region of the Ig portion of the fusion protein may be further modified to reduce immunoglobulin effector function. For example, nucleotide sequences encoding the fusion protein may be modified to encode fusion proteins which replace cysteine residues in the hinge region with serine residues and/or amino acids within the CH2 domain believed to be required for IgC binding to FC receptors and complement activation.

Other Reference Publication (34):
Liu et al., 1989 "Perforin and serine esterase gene expression in stimulated human T cells", J Exp Med 170:2105-2118.

Other Reference Publication (42):
Masson et al., 1986, "Granules of cytotoxic T-lymphocytes contain two serine esterases", EMBO J 5(7):1595-1600.

L11: Entry 9 of 11 File: USPT May 23, 2000

DOCUMENT-IDENTIFIER: US 6066498 A
TITLE: Compositions for the treatment and diagnosis of immune disorders

Detailed Description Text (118):
Described herein are methods for the production of antibodies capable of specifically recognizing one or more differentially expressed or pathway gene product epitopes. Such antibodies can include, but are not limited to, polyclonal antibodies, monoclonal antibodies (mAbs), humanized or chimeric antibodies, single chain antibodies, Fab fragments, F(ab').sub.2 fragments, fragments produced by a Fab expression library, anti-idiotypic (anti-Id) antibodies, and epitope-binding fragments of any of the above. The Ig tails of such antibodies can be modified to reduce complement activation and Fc binding. (See, for example, European Patent No. 239400 B1, Aug. 3, 1994).

Detailed Description Text (420):
The 200 gene product extracellular domain sequence of the Ig-fusion protein consists of amino acid residues 1 to 191 (i.e., the 200 gene product portion ends with the amino acid sequence G-E-T-I-R-T). This 200 gene product extracellular domain is PCR amplified using synthetic oligonucleotides complementary to the 200 gene nucleotide sequence such that the PCR products will include the above 200 gene product amino acid residues 1-191. Further, XhoI and BamHI restriction sites are designed at the 5' and 3' ends of the PCR products, respectively, to facilitate the subsequent insertion into IgG1 expression vectors (See Arurro, A. et al., 1991, Cell 61:1303-1313). In addition to the Ig-region encoded by the IgG1 vectors, the Fc portion of the Ig region can be modified, by amino acid substitutions, to reduce complement activation and Fc binding. (See, e.g., European Patent No. 239400 B1, Aug. 3, 1994).

Other Reference Publication (41):
Liu et al., 1989, "Perforin and serine esterase gene expression in stimulated human T cells", J Exp Med 170:2105-2118.

Other Reference Publication (49):
Masson et al., 1986, "Granules of cytotoxic T-lymphocytes contain two serine esterases", EMBO J 5(7):1595-1600.

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

End of Result Set

Generate Collection Print

L11: Entry 11 of 11 File: USPT Feb 24, 1998

DOCUMENT-IDENTIFIER: US 5721351 A
** See image for Certificate of Correction **
TITLE: Compositions and methods for the treatment and diagnosis of immune disorders

Detailed Description Text (112):
Described herein are methods for the production of antibodies capable of specifically recognizing one or more differentially expressed or pathway gene product epitopes. Such antibodies can include, but are not limited to, polyclonal antibodies, monoclonal antibodies (mAbs), humanized or chimeric antibodies, single chain antibodies, Fab fragments, F(ab').sub.2 fragments, fragments produced by a Fab expression library, anti-idiotypic (anti-Id) antibodies, and epitope-binding fragments of any of the above. The Ig tails of such antibodies can be modified to reduce complement activation and Fc binding. (See, for example, European Patent No. 239400 B1, Aug. 3, 1994).

Detailed Description Text (417):
The 200 gene product extracellular domain sequence of the Ig-fusion protein consists of amino acid residues 1 to 191 (i.e., the 200 gene product portion ends with the amino acid sequence G-E-T-I-R-T). This 200 gene product extracellular domain is PCR amplified using synthetic oligonucleotides complementary to the 200 gene nucleotide sequence such that the PCR products will include the above 200 gene product amino acid residues 1-191. Further, XhoI and BamHI restriction sites are designed at the 5' and 3' ends of the PCR products, respectively, to facilitate the subsequent insertion into IgG1 expression vectors (See Arurro, A. et al., 1991, Cell 61:1303-1313). In addition to the Ig-region encoded by the IgG1 vectors, the Fc portion of the Ig region can be modified, by amino acid substitutions, to reduce complement activation and Fc binding. (See, e.g., European Patent No. 239400 B1, Aug. 3, 1994).

Other Reference Publication (40):
Liu et al., 1989, "Perforin and serine esterase gene expression in stimulated human T cells", J Exp Med 170:2105-2118.

Other Reference Publication (48):
Masson et al., 1986, "Granules of cytotoxic T-lymphocytes contain two serine esterases", EMBO J 5(7):1595-1600.

Previous Doc Next Doc Go to Doc#

L13: Entry 12 of 21 File: USPT Dec 17, 2002

DOCUMENT-IDENTIFIER: US 6495735 B1
TITLE: Transgenic non-human mammals expressing DNA sequences encoding homologous complement restriction factors of a discordant mammalian species

Detailed Description Text (42):
FIG. 5 is a reproduction of the dry gel. Rocket 1 is a negative control containing 50 .mu.l normal human serum (HHS) plus 25 .mu.l VBS including 10 mM EGTA. EGTA is a chelator which removes calcium; calcium is essential for classical pathway complement activation, and so the presence of EGTA ensures that complement can only be activated by the alternative pathway. The left-hand (larger) peak is C3, and the right-hand (smaller) peak is C3bi, a breakdown product of activated C3. In the control, therefore, the small amount of C3bi indicates only a minor amount of complement activation.

First Hit Previous Doc Next Doc Go to Doc#

Generate Collection Print

L13: Entry 20 of 21 File: DWPI Dec 25, 2001

DERWENT-ACC-NO: 2002-113123
DERWENT-WEEK: 200215

TITLE: Administering anti-properdin agents (antibodies) to treat the adverse effects of the alternative and classical complement pathways for the treatment of e.g. ischemia, Parkinson's disease and rheumatoid arthritis

Basic Abstract Text (6):
MECHANISM OF ACTION - Antibody inhibition of properdin; inhibition of the alternative and/or classical complement activation pathways. To test the effect of a blocking anti-properdin monoclonal antibody on inhibition of complement activation in cardiopulmonary bypass (CPB), a tubing loop model of CPB as described by Gong, JR et al., (1996), J. Clin. Immunol. 16:222-229 was utilized. Whole blood from a healthy donor was collected into a 7-ml vacutainer tube containing 20 U of heparin/ml of blood. Polyethylene tubing like that used during CPB (PE 330; I., 2.92 mm; O., 3.73 mm) was filled with 0.5 ml of the heparinized human blood and closed into a loop with a short piece of silicon tubing. Heparinized blood containing 20 mM Ethylene Diamine Tetraacetic Acid (EDTA) (which inactivates complement) served as a background control. Sample and control tubing loops were rotated vertically in a water bath for 1 hour at 37 deg. C.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L14: Entry 3 of 4 File: USPT Mar 30, 1999

DOCUMENT-IDENTIFIER: US 5888519 A
TITLE: Encapsulated high-concentration lipid a compositions as immunogenic agents to produce human antibodies to prevent or treat gram-negative bacterial infections

Brief Summary Text (26):
When lipid A in included in the lipid bilayer it can be demonstrated that anti-lipid A antibodies that are known to have specificities against the lipid headgroup (diglucosamine diphosphate) are readily bound to the liposomal lipid A. This can be shown to cause agglutination or complement fixation, and complement activation can cause lysis of the lipid bilayer resulting in increased permeability of the liposome to marker compounds present in the internal aqueous spaces. Other methods for performing the immunological studies could include using the liposomes containing lipid A as an antigen in enzyme-linked immunosorbent assays, or by using fluorescent antibodies or antibodies labeled with dyes, to "light up" and visualize the occurrence of the immunological reaction at the surface of the liposomes, or by using liposomes as absorbent particles to absorb antibodies. It is concluded from this that the lipid A molecule is oriented in the expected manner with the hydrophilic portion oriented toward the aqueous medium and the hydrophobic (fatty acid) portion buried in the lipid bilayer. Similar types of studies can be used to demonstrate that anti-lipid A antibodies can bind to lipid A that has been absorbed to erythrocytes.

Previous Doc Next Doc Go to Doc#

L15: Entry 29 of 57 File: USPT Mar 2, 1999

DOCUMENT-IDENTIFIER: US 5877396 A
TITLE: Mice mutant for functional Fc receptors and method of treating autoimmune diseases

Detailed Description Text (358):
Other Inflammatory Parameters. Based on the results presented here, Fc receptor engagement by immune complexes is a critical step in the initiation of an antigen-antibody mediated inflammatory response. Since complement activation has been thought to be the key step in the initiation of the Arthus reaction, and since a defective complement system in the -/- mice is an alternative explanation for the results obtained, experiments to document an intact complement system were performed. First, total hemolytic complement levels were determined in -/- and +/+ mice, using sheep RBC's sensitized with rabbit anti-SRBC antibodies as targets (14). Both +/+ and -/- animals had comparable levels of hemolytic complement, averaging 82.+-.30 and 187.+-.76 U/ml, respectively. The ability of the complement cascade to function in vivo was next determined by challenging these animals with intradermal zymosan, which activates complement independent of immune complexes, via the "alternative pathway" (15). As seen in FIG. 20B, right, zymosan induced a dramatic inflammatory response in the -/- mice, which was indistinguishable from that of the +/+ mice (not shown). The data presented in FIG. 20 indicate that inflammatory responses to stimuli which do not interact with Fc receptors, like zymosan or mouse IgG3, are intact in the -/- mice. The defect displayed by these animals in mounting an inflammatory response to IgG immune complexes is therefore most likely the result of deletion of Fc receptors for these immune complexes. The role of complement in the initiation of this response was further assessed by complement depleting +/+ and -/- mice with cobra venom factor (7, 16) and then performing a reverse passive Arthus reaction. As shown in FIGS. 21A and 21B, the +/+ mice show the previously reported attenuation in the inflammatory response, whereas the residual inflammatory response found in the -/- mice was completely ablated, despite the fact that both had levels of hemolytic complement not significantly different from that of heat-inactivated (i.e. complement depleted) serum. From these studies it appears that the role of complement in initiating the immune complex mediated inflammatory response is secondary to that of Fc receptor engagement, although it is undoubtedly necessary for its full expression.

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
Generate Collection Print

L15: Entry 28 of 57 File: USPT Jul 13, 1999

DOCUMENT-IDENTIFIER: US 5922690 A
TITLE: Dermatan disulfate, an inhibitor of thrombin generation and activation

Brief Summary Text (11):
Heparin has also been used to treat complement system abnormalities. The complement system, plays a major role in host defense both through destruction of invading organisms and through mediation of inflammation. Complement abnormalities are unusual conditions characterized by a deficiency or by a dysfunction of any of the more than nineteen normally well-behaved proteins constituting about 10% of the globulins in normal human serum. Patients with complement deficiencies or with complementary dysfunctions also may be susceptible to tissue injury as a result of excessive inflammatory responses. Further, complement activation in the course of recovery from temporary blood vessel occlusion or in response to cardiopulmonary bypass during heart surgery initiates tissue damage beyond that caused by the initial injury. Heparin has been shown to inhibit activity of the alternative, classical and terminal pathways of complement by regulating C1, C1 Inhibitor, C4 binding protein, C3b, factor H and S-protein in a model that predicts in vivo complement inhibition properties. (Edens, R. E., Linhardt, R. J., Bell, C. S., Weiler, J. M., Heparin and Derivatized Heparin Inhibit Zymosan and Cobra Venom Factor Activation of Complement in Serum, Immunopharmacol. 27: 145153 (1994)) However, the anticoagulant activity of heparin contributes to an increased risk of bleeding, electrolyte shifts and thrombocytopenia.

Previous Doc Next Doc Go to Doc#

L15: Entry 27 of 57 File: USPT Sep 28, 1999

DOCUMENT-IDENTIFIER: US 5958457 A
** See image for Certificate of Correction **
TITLE: Compositions for the delivery of antigens

Detailed Description Text (7):
Adjuvants suitable for use in the present invention include, but are not limited to protein carriers such as protein containing appropriate T-cell epitopes; hydrophobic antigens such as proteins with a lipid tail or antigens in oil with added MDP; polyclonal activators of T-cells such as PPD, poly A and poly U; B-cell activators such as antigen-polymerizing factors and B-cell mitogens; macrophage (APC) stimulators such as muramyl dipeptides (MDP) and derivatives thereof; and lipopolysaccharides (LPS); alternate pathway complement activators such as, for example, inulin, zymosan, endotoxin, levamisole, C. parvum; or any combinations thereof. Other useful adjuvants include lipoidal amines in general; polyphophazenes; bacterial toxins such as E-coli heat labile enterotoxin (LT-OA), cholera or diphtheria toxin or subunits, thereof, such as, for example, cholera toxin .beta.-subunit or E-coli heat labile anterotoxin .beta.-subunit; bacterial toxoids; poly or di-saccharides; or any combination thereof such as, for example, cholera toxin and cholera toxin .beta.-subunit.

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
Generate Collection Print

L15: Entry 26 of 57 File: USPT Nov 2, 1999

DOCUMENT-IDENTIFIER: US 5976540 A
TITLE: Compositions comprising complement related proteins and carbohydrates, and methods for producing and using said compositions

Brief Summary Text (45):
Studies of Weisman et al., (1990, Science 249:146-151) have demonstrated that sCR1 can prevent 90% of the generation of C3a and C5a in human serum activated by the yeast cell wall component zymosan. Weisman, et al., (1990, supra) also utilized sCR1 in the rat to inhibit complement activation and reduce the damage due to myocardial infarction. Soluble CR1 also appears to inhibit the complement dependent process of the reverse Arthus reaction (Yeh, et al., 1991, J. Immuno. 146:250-256), and hyperacute xenograft rejection (Pruitt, et al., 1991, Transplantion 52:868-873). Recent data (Moat, et al., 1992, Amer. Rev. Respiratory Disease 145:A845) indicate that sCR1 is of value in preventing complement activation in an experimental model of cardiopulmonary bypass in the pig, a situation where complement activation has been demonstrated.

Previous Doc Next Doc Go to Doc#

L15: Entry 31 of 57 File: USPT Jan 12, 1999

US-PAT-NO: 5859223
DOCUMENT-IDENTIFIER: US 5859223 A
** See image for Certificate of Correction **

TITLE: Soluble CR1 derivatives

DATE-ISSUED: January 12, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mossakowska; Danuta Ewa Irena	Mountfitchet			GB2
Dodd; Ian	Buckland			GB2
Freeman; Anne Mary	Wendover			GB2
Smith; Richard Anthony Godwin	Horseheath			GB2

US-CL-CURRENT: 536/23.5; 435/252.1, 435/252.33, 435/320.1, 435/69.2, 530/380

CLAIMS:

We claim:

1. A DNA molecule encoding a soluble polypeptide comprising no more than three short consensus repeats (SCR) of long homologous repeat A of Complement Receptor 1, wherein the DNA molecule encodes SCR3 and at least one repeat selected from the group consisting of SCR1, SCR2, and SCR4.

2. A DNA molecule encoding a soluble polypeptide comprising no more than one short consensus repeat (SCR) of long homologous repeat A of Complement Receptor 1, wherein the DNA molecule encodes SCR3.

3. A vector comprising a DNA molecule according to claim 1.

4. A vector comprising a DNA molecule according to claim 2.

5. A host cell comprising a DNA molecule according to claim 1.

6. A host cell comprising a DNA molecule according to claim 1.

7. A host cell according to claim 5, wherein the host cell is a bacterium.

8. A host cell according to claim 6, wherein the host cell is a bacterium.

9. A method of producing a soluble polypeptide that can inhibit complement activation, comprising:

expressing in a host cell a DNA molecule encoding a soluble polypeptide

comprising no more than three short consensus repeats (SCR) of long homologous repeat A of Complement Receptor 1, wherein the DNA molecule encodes SCR3 and at least one repeat selected from the group consisting of SCR1, SCR2, and SCR4 to produce the soluble polypeptide; and

harvesting the soluble polypeptide.

10. A method of producing a soluble polypeptide that can inhibit complement activation, comprising:

expressing in a host cell a DNA molecule encoding a soluble polypeptide comprising a DNA molecule encoding a soluble polypeptide comprising no more than one short consensus repeat (SCR) of long homologous repeat A of Complement Receptor 1, wherein the DNA molecule encodes SCR3.

11. A method according to claim 9, wherein the host cell is a bacterium.

12. A method according to claim 10, wherein the host cell is a bacterium.

13. A method according to claim 11, wherein the bacterium is E. coli.

14. A method according to claim 12, wherein the bacterium is E. coli.

Previous Doc Next Doc Go to Doc#

L15: Entry 33 of 57 File: USPT Jan 5, 1999

DOCUMENT-IDENTIFIER: US 5856297 A
TITLE: Human C3b/C4b receptor (CR1)

Detailed Description Text (400):
To determine whether suppression of tissue damage by sCR1 was associated with inhibition of complement activation by ischemic myocardium, another group of buffer-treated (n=7) and sCR1-treated rats (n=8) was subjected to the same ischemia-reperfusion protocol, and the animals were killed 3 hours after reperfusion. The hearts were assessed by nitroblue tetrazolium (NBT) staining (Lillie, R. D., 1965, Histopathologic Technic and Practical Histochemistry, McGraw-Hill, New York ed:3:378) to delineate regions of irreversible injury from viable myocardium, and by immunoperoxidase staining (DeLellis, in Basic Techniques of Immunohistochemistry, DeLellis, Ed., Masson, N.Y., 1981) with a mouse monoclonal antibody to the rat C5b-9 membrane attack complex (Schulze et al., 1989, Kidney Int. 35:60). A mouse peroxidase-antiperoxidase (PAP) system was used for the immunostaining as described. All steps of the procedure were preceded by three 10-minute washings in 0.05M Tris-buffered saline. The sections were fixed in acetone and treated with 0.5 percent H.sub.2 O.sub.2 -methanol solution for 5 minutes, 4 percent heat-inactivated goat serum for 1 hour; and they were then sequentially incubated at room temperature with the primary, mouse monoclonal antibodies at 2 .mu.g/ml for 11 hours, with affinity-purified F(ab').sub.2 goat antiserum to nouse antibody (organon-Tecknika, West Chester, Pa.) for 60 minutes, and with mouse PAP for 60 minutes. The slides were developed with 3,3'-diaminobenzidine tetrahydrochloride and counterstained with Gill's hematoxylin 3. In the NBT-negative, infarcted areas of the control rats (n=7), the C5b-9 complex was present primarily along the endothelium of capillaries and venules, but not in the myocardial fibers. In contrast, in rats that had received sCR1 (n=8), the NBT-negative areas were consistently reduced in size, and little or no C5b-9 complex was detectable in these regions, as exemplified by the representatives sections shown.

L15: Entry 42 of 57 File: USPT Dec 5, 1995

US-PAT-NO: 5472939
DOCUMENT-IDENTIFIER: US 5472939 A

TITLE: Method of treating complement mediated disorders

DATE-ISSUED: December 5, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fearon; Douglas T.	Baltimore	MD		
Klickstein; Lloyd B.	Brookline	MA		
Wong; Winnie W.	Newton	MA		
Carson; Gerald R.	Wellesley	MA		
Concino; Michael F.	Newton	MA		
Ip; Stephen H.	Sudbury	MA		
Makrides; Savvas C.	Bedford	MA		
Marsh, Jr.; Henry C.	Reading	MA		

US-CL-CURRENT: 514/8; 514/12, 514/2, 514/885, 514/886

CLAIMS:

What is claimed is:

1. A method of treating a patient with a complement mediated immune disorder or a disorder which involves undesirable or inappropriate complement activity comprising administering to the patient a protein having an amino acid sequence as depicted in FIGS. 1A to 1P or a fragment thereof comprising at least two short consensus repeats and that has complement regulatory activity.

2. The method according to claim 1 in which the protein or fragment thereof is characterized by the ability to bind C3b.

3. The method according to claim 1 in which the protein or fragment thereof is characterized by factor I cofactor activity, an ability to inhibit C3 convertase activity or an ability to inhibit C5 convertase activity.

4. The method according to claim 1 in which the protein or fragment thereof lacks a transmembrane region.

5. The method according to claim 1 in which the patient is a non-human animal.

6. The method according to claim 1 in which the protein or fragment thereof is characterized by the ability to bind C4b.

7. The method according to claim 1 in which the protein or fragment thereof is

secreted.

8. The method according to claim 1 in which the protein or fragment thereof is expressed as a cell-surface protein.

9. A method of treating a patient with a complement mediated immune disorder or a disorder which involves undesirable or inappropriate complement activity comprising administering to the patient a CR1 molecule comprising at least two short consensus repeats and that substantially lacks a transmembrane region and that has complement regulatory activity.

10. The method according to claim 9 in which the CR1 molecule is secreted by a cell in which it is expressed.

11. The method according to claim 9 in which the patient is a non-human animal.

12. A method of treating or preventing damage in a patient caused by a myocardial infarct comprising administering to the patient a protein having an amino acid sequence as depicted in FIGS. 1A to 1P or a fragment thereof comprising at least two short consensus repeats and that has complement regulatory activity.

13. The method according to claim 12 in which the patient is a non-human animal.

14. A method of treating or preventing damage in a patient caused by a myocardial infarct comprising administering to the patient a CR1 molecule comprising at least two short consensus repeats and that substantially lacks a transmembrane region and that has complement regulatory activity.

15. The method according to claim 14 in which the CR1 molecule is secreted by a cell in which it is expressed.

16. The method according to claim 14 in which the CR1 molecule is functional as detected by the ability in vitro to inhibit neutrophil oxidative burst, complement-mediated hemolysis, or C3a and C5a production.

17. The method according to claim 14 in which the molecule is encoded by a nucleic acid vector selected from the group consisting of pT-CR1c1, PT-CR1c2, pT-CR1c3, pT-CR1c4, and pT-CR1c5.

18. The method according to claim 14 in which the patient is a non-human animal.

19. A method of treating or preventing damage in a patient caused by inflammation comprising administering to the patient a protein having an amino acid sequence as depicted in FIGS. 1A to 1P or a fragment thereof comprising at least two short consensus repeats and that has complement regulatory activity.

20. The method according to claim 19 in which the patient is a non-human animal.

21. A method of treating or preventing damage in a patient caused by inflammation comprising administering to the patient a CR1 molecule comprising

at least two short consensus repeats and that substantially lacks a transmembrane region and that has complement regulatory activity.

22. The method according to claim 21 in which the CR1 molecule is secreted by a cell in which it is expressed.

23. The method according to claim 21 in which the molecule is functional as detected by the ability in vitro to inhibit neutrophil oxidative burst, complement-mediated hemolysis, or C3a and C5a production.

24. The method according to claim 21 in which the molecule is encoded by a nucleic acid vector selected from the group consisting of pT-CR1c1, pT-CR1c2, pT-CR1c3, pT-CR1c4, and pT-CR1c5.

25. The method according to claim 21 in which the patient is a non-human animal.

26. A method of treating a patient with a complement mediated immune disorder or a disorder which involves undesirable or inappropriate complement activity comprising administering to the patient a CR1 molecule that comprises LHR-A, LHR-B, LHR-C and LHR-D, and that substantially lacks a transmembrane region and a cytoplasmic region, and that has complement regulatory activity.

27. The method according to claim 26 in which the CR1 molecule is encoded by a nucleic acid vector selected from the group consisting of pBSCR1c, pBSCR1s, pBMCR1c, pBSCR1c/pTCSgpt, and pBSCR1s/pTCSgpt.

28. The method according to claim 27 in which the CR1 molecule is expressed by a Chinese hamster ovary cell DUX B11, carrying plasmid pBSCR1c/pTCSgpt, as deposited with the ATCC and assigned accession number CRL 10052.

29. A method of treating or preventing damage in a patient caused by a myocardial infarct comprising administering to the patient a CR1 molecule that comprises LHR-A, LHR-B, LHR-C and LHR-D, and that substantially lacks a transmembrane region and a cytoplasmic region, and that has complement regulatory activity.

30. The method according to claim 29 in which the CR1 molecule is encoded by a nucleic acid vector selected from the group consisting of pBSCR1c, pBSCR1s, pBMCR1c, pBSCR1c/pTCSgpt, and pBSCR1s/pTCSgpt.

31. The method according to claim 30 in which the CR1 molecule is expressed by a Chinese hamster ovary cell DUX B11, carrying plasmid pBSCR1c/pTCSgpt, as deposited with the ATCC and assigned accession number CRL 10052.

32. A method of treating or preventing damage in a patient caused by inflammation comprising administering to the patient a CR1 molecule that comprises LHR-A, LHR-B, LHR-C and LHR-D, and that substantially lacks a transmembrane region and a cytoplasmic region, and that has complement regulatory activity.

33. The method according to claim 32 in which the CR1 molecule is encoded by a nucleic acid vector selected from the group consisting of pBSCR1c, pBSCR1s, pBMCR1c, pBSCR1c/pTCSgpt, and pBSCR1s/pTCSgpt.

34. The method according to claim 33 in which the CR1 molecule is expressed by a Chinese hamster ovary cell DUX B11, carrying plasmid pBSCR1c/pTCSgpt, as

deposited with the ATCC and assigned accession number CRL 10052.

35. A method of treating a patient with a complement mediated immune disorder or a disorder which involves undesirable or inappropriate complement activity comprising administering to the patient a CR1 molecule that comprises LHR-B, LHR-C and LHR-D, and lacks LHR-A, and that substantially lacks a transmembrane region and a cytoplasmic region, and that has complement regulatory activity.

36. A method of treating or preventing damage in a patient caused by a myocardial infarct comprising administering to the patient a CR1 molecule that comprises LHR-B, LHR-C and LHR-D, and lacks LHR-A, and that substantially lacks a transmembrane region and a cytoplasmic intracellular region, and that has complement regulatory activity.

37. A method of treating or preventing damage in a patient caused by inflammation comprising administering to the patient a CR1 molecule that comprises LHR-B, LHR-C and LHR-D, and lacks LHR-A, and that substantially lacks a transmembrane region and a cytoplasmic region, and that has complement regulatory activity.

Previous Doc Next Doc Go to Doc#

L15: Entry 51 of 57 File: USPT Oct 13, 1987

US-PAT-NO: 4699783
DOCUMENT-IDENTIFIER: US 4699783 A

TITLE: Products and methods for treatment of cancer

DATE-ISSUED: October 13, 1987

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Terman; David S.	Carmel	CA	93923	
Balint; Joseph P.	Clifton	NJ	07011	
Langone; John J.	Houston	TX	77071	

US-CL-CURRENT: 424/178.1; 530/389.7, 530/413, 530/419, 530/421

CLAIMS:

What is claimed is:

1. A complex of protein A-IgG having the following properties,

(a) comprises predominantly IgG heavy and light chains and protein A by polyacrylamide gel electrophoresis,

(b) is identified in heavy sedimenting 7S or >7S fractions on sucrose density gradient with increased Clq binding activity,

(c) dissociates into lower molecular weight Clq binding fragments under acid conditions,

(d) is precipitable with 5% polyethylene glycol or other imunoglobulin precipating procedures known to those skilled in the art,

(e) has anti-complementary activity,

(f) inhibits Fc dependent lymphocyte rosette formation,

(g) induces neutrophils to aggregate and release myeloperoxidase, cathepsins and superoxide anions,

(h) induces hemagglutination of canine erythrocytes,

(i) induces complement activation with generation of anaphylatoxins,

(j) induces non-specific complement dependent cytotoxicity of canine mammary adenocarcinoma cells, murine L cells and human erythrocytes, and

(k) is generated by interaction of plasma with either free or non-covalently bound protein A by complement independent mechanism and is not present in pretreatment serum.

2. A complex of protein A-IgG having the following properties,

(a) comprises predominantly IgG heavy and light chains and protein A by polyacrylamide gel electrophoresis,

(b) is identified in heavy sedimenting 7S or >7S fractions on sucrose density gradient with increased Clq binding activity,

(c) dissociates into lower molecular weight Clq binding fragments under acid conditions,

(d) is precipitable with 5% polyethylene glycol or other procedures for precipitating immunoglobulins known to those skilled in the art,

(e) has anti-complementary activity,

(f) inhibits Fc dependent lymphocyte rosette formation,

(g) induces neutrophils to release myeloperoxidase, cathepsins and superoxide anions,

(h) induces hemagglutination of canine erythrocytes,

(i) contains antibodies which retain their antigen affinity and have relatively increased cytotoxic capabilitites in the presence of complement, and

(j) when infused into tumor bearing hosts at various molar ratios of protein A-IgG complexes results in tumoricidal reactions and tumor regressions.

3. A method of producing the complex of claim 1 comprising,

isolating the protein A-IgG complex from one of tumor bearing and normal plasma after perfusion over immobilized staphylococcus aureus protein A by precipitating the protein A-IgG complex from the perfused plasma.

4. A method of producing the complex of claim 2 comprising,

incubating protein A with IgG.

5. A method of producing the complex of claim 1 comprising,

incubating protein A with IgG, and

isolating the resulting protein A-IgG complex.

6. The method of claim 5 wherein,

the IgG has tumor specificity.

7. The complex of claim 1 where,

the protein A used in preparing the protein A-IgG preparation includes staphylococcal enterotoxins.

8. The complex of claim 1 where,

the protein A used in preparing the protein A-IgG complex includes staphylococcal enterotoxins A, B, C, E, and F.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#
End of Result Set

Generate Collection Print

L20: Entry 1 of 1 File: USPT Nov 18, 1997

DOCUMENT-IDENTIFIER: US 5688501 A
TITLE: Antibacterial therapy with bacteriophage genotypically modified to delay inactivation by the host defense system

Brief Summary Text (33):
Complement components fix to bacteriophages, and these bacteriophages then adhere to certain white blood cells (such as macrophages) that express complement receptors. Numerous peptides have been synthesized that antagonize the functions of the various complement components. [See e.g. Lambris, J. D. et al, "Use of synthetic peptides in exploring and modifying complement reactivities" in Activators and Inhibitors of Complement, ed. R. Sim, Kluwer Academic Publishers, Boston, 1993.] Lambris et al. (op.cit.) cite "a series of synthetic peptides spanning the covertase cleavage site in C3 (that are) found to inhibit complement activation by both the classical and alternative pathways". Among the peptides cited is a six amino acid peptide residues 746-751 of C3) that "inhibits both pathways equally well".

Other Reference Publication (41):
Shayegani et al., "Correlation of Staphylococci-Induced Delayed-Type Hypersensitivy and Nonspecific Resistance During Their Development, Long-Term Duration and Adoptive Transfer", Jour. of the Reticu. Soc., vol. 28, No. 3, pp. 265-274, 1980.

Previous Doc Next Doc Go to Doc#

Refine Search

Search Results -

Terms	Documents
L16 and (hypersensitivy)	1

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L20

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Thursday, June 29, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L20	L16 and (hypersensitivy)	1	L20
L19	L16 and (cremophor)	0	L19
L18	L16 and (polyethoxylat$)	1	L18
L17	L16 and (taxol or paclitaxel or cyclosporin or doxorubicin or polynucleotide or hemoglobin)	30	L17
L16	(complement adj3 activat$ adj3 inhibit$)	118	L16
L15	(complement adj3 activat$) same (zymosan or pap or polyanion or indel$)	57	L15
L14	(complement adj3 activat$) same (anti$lipid)	4	L14
L13	(complement adj3 activat$) same (indomethacin or egta or complestatin or diamine)	21	L13
L12	(complement adj3 activat$) and (indomethacin or egta or complestatin or diamine)	343	L12

L11	(complement adj3 activat$) and (serine adj1 esterase)	11	L11
L10	(compliment adj3 activat$) and (antibod$)	8	L10
L9	(compliment adj3 activat$) and (serine adj1 esterase)	0	L9
L8	(compliment adj3 activat$) and (indomethacin or egta or complestatin or diamine)	0	L8
L7	(compliment ajd3 activat$) and (indomethacin or egta or complestatin or diamine)	31335	L7
L6	compliment$ adj3 activat$	44	L6
L5	L3 and (indomethacin or egta or complestatin or diamine)	1	L5
L4	L3 and (serine adj1 esterase)	0	L4
L3	compliment$ adj3 inhibit$	34	L3
L2	compliment adj2 inhibit$	11	L2
L1	compliment adj1 activation adj1 inhibit$	0	L1

END OF SEARCH HISTORY

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L3: Entry 4 of 12 File: USPT Sep 11, 2001

DOCUMENT-IDENTIFIER: US 6287558 B1
TITLE: Devices containing cells or tissue and an agent that inhibits damage by a host cell molecule

Detailed Description Text (14):
Additional rescue agents of protein or peptide nature include: a naturally-occuring or an engineered cytokine binding protein, such as TNF BP-I; an antibody to an inflammation-related enzyme, such as anti-host neutrophil myeloperoxidase; an engineered inhibitory binding protein to a host enzyme; a naturally occurring complement inhibitor such as Factor H or a soluble form of CDAF; an engineered complement binding-protein or inhibitor; or an enzyme that neutralizes an inflammatory substance, such as superoxide dismutase which reduces a superoxide anion.

Previous Doc Next Doc Go to Doc#

L3: Entry 7 of 12 File: USPT Apr 24, 2001

DOCUMENT-IDENTIFIER: US 6221657 B1
TITLE: Modified human C3 DNA sequences and vectors

Brief Summary Text (8):
Existing approaches to preventing complement-mediated damage have targeted the use of down-regulatory proteins (CR1, MCP, DAF and factors H and I) to inhibit complement activation. Complement inhibitors like factor I, factor H and soluble derivatives of the membrane-bound proteins CR1, DAF, MCP do suppress the fluid-phase amplification loop of the alternative pathway. Therefore there have been attempts to use these molecules, particularly CR1 (which seems to be the most potent) to reduce complement-mediated damage in models of physiological situations [10, 18].

First Hit Previous Doc Next Doc Go to Doc#

Generate Collection Print

L3: Entry 11 of 12 File: EPAB Jul 21, 1994

DOCUMENT-IDENTIFIER: WO 9416062 A1
TITLE: COMPLEMENT REGULATORY PROTEINS OF HERPESVIRUS SAIMIRI

Abstract Text (1):
Gene sequences for three complement regulatory proteins encoded within the genome of Herpesvirus Saimiri (HVS) are disclosed, namely, mCCPH, sCCPH, and HVS-15. mCCPH and sCCPH share substantial homology with the human complement inhibitory proteins factor H, CD35, CD46, CD55, and C4bp which inhibit C3 convertase activity in the complement cascade. HVS-15 shares substantial homology with the human complement inhibitory protein CD59 which inhibits formation of the membrane attack complex of the complement system. The gene sequences and corresponding proteins can be used as therapeutic agents to control the complement arm of the immune system.

Previous Doc Next Doc Go to Doc#

Refine Search

Search Results -

Terms	Documents
(complement adj3 inhibit$) adj5 (factor adj1 h)	12

Database: US Pre-Grant Publication Full-Text Database; US Patents Full-Text Database; US OCR Full-Text Database; EPO Abstracts Database; JPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L3

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Thursday, June 29, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR			
L3	(complement adj3 inhibit$) adj5 (factor adj1 h)	12	L3
L2	(complement adj3 inhibit$) adj5 factor	79	L2
L1	(complement adj3 inhibit$) same factor	343	L1

END OF SEARCH HISTORY

First Hit Previous Doc Next Doc Go to Doc#

Generate Collection Print

L3: Entry 10 of 12 File: JPAB Jun 3, 1994

DOCUMENT-IDENTIFIER: JP 06157342 A
TITLE: USE OF COMPLEMENT INHIBITOR FOR PRODUCTION OF MEDICINE FOR PREVENTION AND TREATMENT OF INFLAMMATORY INTESTINE, DERMATOSIS AND PURPURA

Abstract Text (2):
CONSTITUTION: This medicine is the complement inhibitor and particularly its C1 inactivator and I factor or H factor has an action to suppress Arthus reaction. The soln. contg. such C1 inactivator and I factor or H factor is useful for prevention and treatment of the inflammatory dermatosis; for example, pustular skin disease, dermatitis group, psoriasis, intestinal disease, particularly, Chrohn's disease, tumorous colon inflammation and inflammatory purpura. The soln. allows intravenous, intramuscular or subcutaneous administration and the dosage thereof is the C1 inactivator: 1 to 500 IU/kg/day, the I factor: 0.05 to 100 mg/BW/day, the H factor: 0.005 to 100 mg/kg/BW/day. The separate administration of these inhibitors or the administration in combination thereof is possible.

Previous Doc Next Doc Go to Doc#

First Hit Fwd Refs Previous Doc Next Doc Go to Doc#

Generate Collection Print

L4: Entry 19 of 22 File: USPT Dec 26, 1995

DOCUMENT-IDENTIFIER: US 5478860 A
TITLE: Stable microemulsions for hydrophobic compound delivery

Brief Summary Text (6):
Taxol is formulated in cremophor EL, a common lipid emulsion used for hydrophobic drugs. Cremophor EL is polyoxyethylated castor oil in anhydrous ethanol (50:50). It is well established, however, that the solubilizing agent cremophor EL can promote acute toxic reactions typically expressed as hypersensitivity (Lassus et al., Proc. Am. Soc. Oncol. 4:268, 1985). At present, this is managed clinically though the use of premedication regimes with anti-inflammatory agents such as corticosteroids, antihistamines, dexamethasone and diphenhydramine. Even with premedication, 41% of all patients will exhibit a hypersensitivity reaction (Taxol package insert from Bristol-Myers Squibb; also, Kris et al., Cancer Treatment Reports 70(5):605, 1986). In order to reduce acute reactions to the present taxol formulation, the drug has to be given by intravenous infusion, typically over 24 hours. This step adds significantly to the cost of patient care.

Previous Doc Next Doc Go to Doc#